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#18	Search **SDF-1 antibody and vascularization**	12:02:43	4
#16	Search **SDF-1 antibody and cancer**	11:59:28	34
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☐	L7	L6 AND cancer	15
☐	L6	(SDF-1 OR SDF1 OR CXCL12) NEAR antibody	20
☐	L5	(Murakami)[IN] AND CXCR4	1
☐	L4	(Bertolini OR Dell OR Martinelli OR pruneri)[IN] AND SDF-1	0
☐	L3	(Bertolini OR Dell OR Martinelli OR pruneri)[IN] AND CXCR4	0
☐	L2	L1 AND cancer	254
☐	L1	((CXCR4 OR CXCR-4)NEAR antibody) AND vascularization	255

END OF SEARCH HISTORY

Symbol	Name	Synonyms	Organism
CXCR4	chemokine (C-X-C motif) receptor 4	CD184, CD184 antigen, C-X-C chemokine receptor type 4, CXCR-4, CXC-R4, D2S201E, FB22, fusin, Fusin, HM89, HSY3RR, LAP3, LCR1, LESTR, Leukocyte-derived seven transmembrane domain receptor, NPY3R, NPYR, NPYRL, NPYY3R, SDF-1 receptor, Stromal cell-derived factor 1 receptor, WHIM	Homo sapiens

UniProt	P61073, Q53S69, Q5MIL4
IntAct	P61073
OMIM	193670, 162643
NCBI Gene	7852
NCBI RefSeq	NP_003458, NP_001008540
NCBI RefSeq	NM_003467, NM_001008540
NCBI UniGene	7852
NCBI Accession	CR601301, AF025375

Homologues of CXCR4 ...

Interaction information for this gene ...

Breaking news for this gene ... new

Enhanced PubMed/Google query ...

These results indicate that the increased turnover of CD8+ T cells in HIV-infected subjects is mediated by the HIV envelope protein through the **CXCR4** chemokine receptor.

AMD3100 was shown to interact with the CXC-chemokine receptor **CXCR4**, the main coreceptor used by T-cell tropic strains of HIV.

RESULTS: O-sulfated [K5-OS(H)] and N,O-sulfated [K5-N,OS(H)] K5 derivatives with high degree of sulfation inhibited the replication of an HIV strain using **CXCR4** as entry co-receptor (X4 virus) in both cell lines and T-cell blasts.

Recently, the alpha-chemokine receptor **CXCR4** has been reported to mediate apoptosis in neuronal cells and in CD4(+) and CD8(+) T cells after its binding to HIV-1 envelope proteins.

We demonstrate that CD4+ T cell trafficking in transgenic mice is biased toward bone marrow essentially due to **CXCR4** overexpression, resulting in the severe loss of CD4+ T cells from circulating blood.

Based on these findings, we propose a hypothetical model in which the dual function of **CXCR4** in HIV-1 infection and in lymphocyte trafficking may cooperatively induce progressive HIV-1 infection and CD4+ T cell decline in patients.

Progressive and persistent downregulation of surface **CXCR4** in CD4(+) T cells infected with human herpesvirus 7.

E913 was inactive against T cell tropic (X4) HIV-1; however, when combined with a **CXCR4** antagonist AMD-3100, E913 potently and synergistically inhibited the replication of dualtropic HIV-1 and a 50:50 mixture of R5 and X4 HIV-1.

It has been established that HIV envelope (Env) glycoprotein mediates T cell loss via a mechanism that requires **CXCR4** binding.

We sought to better characterize the effects of HIV Nef on T cell function by examining chemotaxis in response to stromal cell-derived factor-

1alpha (SDF-1alpha) as well as **CXCR4** signaling molecules.

Multi-color flow cytometric analysis on human CD8(+) T cell subsets revealed that **CXCR4** is predominantly expressed on CD8(+) T cells with the naive CD27(+)CD28(+)CD45RA(+) phenotype, and is down-regulated during differentiation into those with an effector phenotype.

Furthermore, up-regulation of **CXCR4** was also associated with the enhancement of HIV replication in human CD4+ T lymphocytes.

Co-culture of monocytes with purified CD3+ T cells led to enhanced basal expression of **CXCR4** on monocytes.

Raised plasma levels of stromal cell-derived factor (SDF)-1alpha and interleukin (IL)-7, cytokines important in T cell development, and in the modulation of surface **CXCR4** expression, have been reported to be associated with HIV-1 disease progression.

X4-tropic HIV1 strains, which use CD4 and **CXCR4** as receptors for cell entry, caused death of unstimulated noncycling primary CD4+ T cells only if the viruses were produced by dying, productively infected T cells, but not by living, chronically infected T cells or by living HIV1-transfected HeLa cells.

To elucidate a possible mechanism, we determined that cholesterol extraction by hydroxypropyl-beta-cyclodextrin (BCD) inhibits stromal cell-derived factor 1alpha (SDF-1alpha) binding to **CXCR4** on T cell lines and PBMCs.

Here we report that IL-4 specifically enhances cell surface expression of **CXCR4** on resting peripheral and cord blood T cells.

This effect is not due either to the amount of circulating virus or to the replication kinetics of those strains, but rather depends on the ability of T-tropic viruses to infect T-cell precursors using **CXCR4** receptors, which are highly expressed in immature thymocytes.

We have isolated a homolog of **hCXCR-4** from a murine T-cell cDNA library and have examined its ability to function as an HIV-1 coreceptor. mCXCR-4 was found to be 91% identical to the human receptor at the amino acid level, with sequence differences concentrated in extracellular domains.

In vivo evolution of human immunodeficiency virus type 1 toward increased pathogenicity through **CXCR4**-mediated killing of uninfected CD4 T cells.

In the thymus, a large number of immature and mature T lymphocytes express **CXCR4**, which may render these cells susceptible to infection by syncytium-inducing viral variants that use this coreceptor for entry.

CXCR4 mRNA expression was identified in infiltrating neointimal T lymphocytes, but not smooth muscle cells by immunolabeling.

Importantly, antibody against **CXCR4** or a neutralizing antibody against HIV-1 gp120 V3 loop blocks T-cell tropic HIV-1 entry into HT-29 cells.

We have generated transgenic mice predominantly expressing human CD4 and **CXCR4** on their CD4-positive T lymphocytes (CD4+ T cells).

Autophagy is involved in T cell death after binding of HIV-1 envelope proteins to **CXCR4**.

CXCR4 utilization is sufficient to trigger CD4+ T cell depletion in HIV-1-infected human lymphoid tissue.

CXCR4 activation in CD4 T cells by SDF1alpha led to the activation of the prosurvival second messengers, Akt and extracellular signal-regulated protein kinase.

When primary activated CD4(+) T cells acutely infected with HIV-1(NL4-3) (**CXCR4**-using T-cell-line-tropic) were cocultured with PFA-fixed gp34(+) human T-cell leukemia virus type 1-bearing MT-2 cells or SV-T2/gp34 cells, HIV-1 production was also markedly enhanced.

Lysophosphatidylcholine up-regulates **CXCR4** chemokine receptor expression in human CD4 T cells.

[FEBS Lett. (1998) 426, 367-372], using a virus with an identical V3 region, suggested that elimination of this particular glycan reduced the ability of T-tropic HIV to bind to **CXCR4** and hence its ability to infect T cell lines.

The binding of HIV-derived recombinant soluble (s)gp120 to the CD4(+)/CXCR4(+) A3.01 T cell line inhibits the binding of the **CXCR4**-specific monoclonal antibodies 12G5, which interacts with the second extracellular loop, and 6H8, which binds the NH2 terminus.

Cortisol-induced **CXCR4** augmentation mobilizes T lymphocytes after acute physical stress.

DPP also regulates an extensive series of genes under the control of protein kinase C, including several involved in T cell activation and cytoskeleton reorganization, and represses expression of the HIV-1 receptor CD4 and coreceptor **CXCR4**.

Finally, a significant positive correlation among the proportions of circulating **CXCR4**-expressing CD4+ T cells, plasma viremia, and the probability to isolate SI strains was found.

Increased expression of HIV co-receptor **CXCR4** on CD4+ T-cells in patients with active visceral leishmaniasis.

In the presence of monocyte-derived macrophages, **CXCR4**-mediated apoptosis targeted mostly CD8(+) T cells, with CD4(+) T cells being more weakly affected.

Inhibition of **CXCR4**-tropic HIV-1 infection by lipopolysaccharide: evidence of different mechanisms in macrophages and T lymphocytes.

TCR-mediated activation of allergen-specific CD45RO(+) memory T lymphocytes results in down-regulation of cell-surface **CXCR4** expression and a strongly reduced capacity to migrate in response to stromal cell-derived factor-1.

Overall up-regulation of **CXCR4** coreceptor on the GC B cells and the CD4(+) T cells surrounding the GC provides the predominant replication and acquisition of the newly formed X4 HIV-1 variants.

CXCR4 usage was conferred by a minimum of two arginine substitutions, regardless of combination, whereas arginine substitutions at position 8 and 11 were required for T-cell line tropism.

CXCR4 expression on monocytes is up-regulated by dexamethasone and is modulated by autologous CD3+ T cells.

Taken together, these results show that common gamma-chain-interacting cytokines as well as signals mediated via noncognate interactions between activated dendritic cells and memory T cells are involved in the up-regulation of **CXCR4** expression.

CXCR4 expression was significantly higher in liver metastases (n = 39) compared with primary CRC tumors (n = 100; P < .0001).

These results suggest that HIV-infected cells can induce autophagy in bystander CD4+ T lymphocytes through contact of Env with **CXCR4**, leading to apoptotic cell death, a mechanism most likely contributing to immunodeficiency.

We also define the kinetics of the HIV life cycle in primary activated human CD4(+)-enriched T cells by using an HIV-1 reporter virus system pseudotyped with the **CXCR4**-dependent HIV-1 envelope gene of NL4-3.

In contrast, SDF-1alpha binds to two affinity states of **CXCR4** in T-cell membranes, which are modulated by guanine nucleotides.

Both CD4(+) and CD8(+) T cell apoptosis could be inhibited by **CXCR4** blockade, mostly in acquired immunodeficiency syndrome subjects and more weakly in asymptomatic HIV-positive subjects, and depended only partially on the syncytium-inducing/non-syncytium-inducing viral envelope phenotype.

Follicular dendritic cell regulation of **CXCR4**-mediated germinal center CD4 T cell migration.

Thus exercise-elicited endogenous cortisol effectively augments **CXCR4** expression on T lymphocytes, which may account for lymphopenia after exercise.

In contrast to earlier findings in mucosal models such as human skin, we demonstrate that the majority of T cells and macrophages but none or few dendritic cells (DC) express the HIV-1 coreceptor CCR5 in normal human cervicovaginal mucosa, whereas all three cell types express the coreceptor **CXCR4**.

RESULTS: Overall, the **CXCR4** promoter exhibited the highest luciferase activity in breast cancer cell lines, primary breast cancer cells

and **breast cancer** tissue slices.

BACKGROUND: The CXC chemokine receptor 4 (**CXCR4**) is predominantly expressed on inactivated naive **T lymphocytes**, **B lymphocytes**, **dendritic cells**, and **endothelial cells**.

CONCLUSIONS: These findings indicate that **CXCR4** is expressed and active in human **melanoma metastases**, suggesting that active inhibitors such as **AMD3100** may be experienced in human **melanoma**.

The **CXCR4 [?]** antagonist, **AMD3100**, stimulates a rapid increase in circulating numbers of haematopoeitic **progenitor cells** (HPCs) in both mice and human healthy volunteers.

We have investigated the expression of the CXC chemokine receptor 4 (**CXCR4**) on primary cultures of type II alveolar **epithelial cells**, their transformed counterpart, the A549 **cell line** and also on other **epithelial cell** lines from various tissues.

MATERIALS AND METHODS: The expression of **CXCR4** was evaluated by **immunohistochemistry** of colorectal tissue samples and by **flow cytometry** on Caco2, GEO, SW480, SW48, Lovo and SW620 human colon carcinoma **cell lines**.

Also, **AMD3100** inhibited stromal cell-derived factor (SDF)-1-induced **endocytosis** of **CXCR4**, but did not affect phorbol ester-induced receptor internalization.

Topical glucocorticoid therapy directly induces **up-regulation** of functional **CXCR4** on primed **T lymphocytes** in the **aqueous humor** of patients with **uveitis**.

In addition, following **coculture** with cells expressing **gp120**, a Fas-independent **apoptosis** involving **mitochondria** and caspase activation is also observed in primary **umbilical cord** blood CD4(+) **T lymphocytes** expressing high levels of **CXCR4**.

Syncytia formation could be blocked by **CXCR4** antagonist **AMD3100**, establishing the importance of this receptor in **FIV** gp120 binding.

It is concluded that **AMD3100** acts on the **CXCR4** receptor through binding to Asp(171) in TM-IV and Asp(262) in TM-VI with each of its **cyclam** moieties, and it is suggested that part of its function is associated with a conformational constraint imposed upon the receptor by the connecting phenylenebismethylene linker.

We confirmed these effects in the human **neuroblastoma cell line** SH-SY5Y, which endogenously expresses **CXCR4**.

BACKGROUND: **AMD3100**, a selective antagonist of **CXCR4**, rapidly mobilizes CD34+ **hematopoietic progenitor cells** (HPCs) from marrow to peripheral blood with minimal side effects.

Both primary tumors and **lymph node metastases** exhibited higher levels of **CXCR4** expression compared to non-neoplastic breast tissues.

Overexpression of **CXCR-4** in **glioblastoma cell lines** enhanced their soft agar colony-forming capability.

The amino acids in **CXCR4** necessary for interaction with an inverse agonist, T140, and a weak partial agonist, **AMD3100**, identified by **alanine** scanning mutants, were spatially consistent when computationally docked.

The **T-cell** (T)-tropic HXB2-based virus, which utilizes **CXCR4** as the entry coreceptor, carrying a **Cys**-to-Ser mutation at residue 764 or 837 or at both replicated with wild-type (WT) **virus replication kinetics** in CD4+ **T cells**.

Also, **AMD3100**, a specific **CXCR4** antagonist with potent antiviral activity against T-tropic **HIV** strains (**50% inhibitory concentration** ¿IC (50), 1 to 10 ng/ml), completely failed to inhibit **HHV-7** infection (IC(50), >250 microg/ml).

The results showed that the levels of functional **CXCR4** expression at both mRNA and protein levels by several human **glioma cell lines** were correlated with the degree of differentiation of the tumor cells.

To test this, levels of **CXCR4** expression were determined for several human **prostate cancer cell lines** by reverse transcription-PCR and **Western blotting**.

A point mutation that confers constitutive activity to CXCR4 reveals that T140 is an inverse agonist and that AMD3100 and ALX40-4C are weak partial agonists.

Insight into the mechanism for CXCR4 antagonists will allow for the development of a new generation of agents that lack partial agonist activity that may induce toxicities, as observed for AMD3100.

CXCR4 expression in osteosarcoma cell lines and tumor samples: evidence for expression by tumor cells.

The interaction of the CXCR4 antagonist AMD3100 with its target is greatly influenced by specific aspartate residues in the receptor protein, including Asp(171) and Asp(262).

Safety, pharmacokinetics, and antiviral activity of AMD3100, a selective CXCR4 receptor inhibitor, in HIV-1 infection.

The normal primary cultured thyroid cells and ATC cell lines expressed CXCR4 and stromal cell-derived factor (SDF)-1 alpha transcripts, detected by RT-PCR.

Importantly, the small-molecule CXCR4-specific inhibitor, 4F-Benzoyl-TE14011 (T140), effectively blocked osteoclast formation stimulated by the myeloma cell line, RPMI-8226.

Therefore, we analysed CXCL12alpha/CXCR4 expression and function in four human kidney cancer cell lines (A-498, CAKI-1, CAKI-2, HA-7), 10 freshly harvested human tumour samples and corresponding normal kidney tissue.

We demonstrated that inhibitors of histone deacetylases, currently being tested in clinical trials for the treatment of various tumours, extensively downregulated CXCR4 protein and mRNA levels in leukaemia cell lines and lymphoblasts from patients with childhood acute leukaemia.

We demonstrate that Ewing's sarcoma tumors as well as Ewing's sarcoma cell lines predominantly express the CXCR4 chemokine receptor.

All B-lymphoma cells freshly isolated from these patients and most laboratory B-lymphoma cell lines, including follicular, diffuse large, and Burkitt's lymphoma cells, expressed surface CXCR4 and migrated in the presence of recombinant human SDF-1alpha.

The luciferase activities in multiple cancer cell lines infected with recombinant adenovirus reAdGL3BCXCR4 or the control vector reAdGL3BCMV revealed that the CXCR4 promoter exhibited relatively high transcriptional activity in a breast cancer cell line, MDA-MB-361, and two ovarian cancer cell lines, OVCAR-3 and SKOV3. ip1, 65% (P=0.0087), 16.7% (P=0.1) and 20% (P=0.0079) compared to that of the CMV promoter, respectively, and low expression, 4.9 and 0.1%, respectively, in both normal cell lines HFBC and HMEC.

The expression of CXCR4 was detected in six pancreatic cancer cell lines by Western blotting and immunocytochemistry.

In this regard, tumor cell migration and metastasis have recently been shown to be regulated by chemokines and their respective receptors (e.g., SDF-1alpha/CXCR4).

CXCR4 gene expression in primary CRC demonstrated significant associations with recurrence, survival, and liver metastasis.

This chemotaxis was inhibited by Pertussis toxin, which uncouples Gi proteins and the bicyclam AMD3100, a highly selective CXCR4 antagonist, as well as by an inhibitor of the MAP kinase pathway.

In contrast, AMD3100 potently inhibited CXCR4-mediated calcium signaling and chemotaxis in a concentration-dependent manner in different cell types.

CXCR4 [?]-low-expressing MCF-7 formed small tumor at inoculated site in SCID mice 8-9 weeks after inoculation while completely failed to metastasis into various organs.

CXC chemokine receptor 4 (CXCR4) plays a role in the development of immune and central nervous systems as well as in cancer growth and metastasis.

These properties identify **CXCR4** as a potential target for the attenuation of **bladder cancer metastases**.

CXCR4 expression is associated with **lymph-node metastasis** of oral **squamous cell carcinoma**.

There was a significant inverse correlation between **methylation** and mRNA expression level of **CXCR4** (P=0.008) in a large panel of **pancreatic cancer cell lines**.

Treatment of **glioblastoma cell lines** with **CXCR-4** and SDFbeta-1 specific antibodies caused inhibition of **glioblastoma cell proliferation**.

RESULTS: High **CXCR4** expression in tumor specimens (n = 57) from AJCC stage I/II patients was associated with increased risk for local recurrence and/or distant **metastasis** (**risk ratio**, 1.35; 95% CI, 1.09 to 1.68; P = .0065).

The non-peptide **CXCR4** receptor antagonist **AMD3100**, which is a potent blocker of **human immunodeficiency virus** cell entry, is a symmetrical **bicyclam** composed of two identical **1,4,8,11-tetraazacyclotetradecane** (**cyclam**) moieties connected by a relatively rigid phenylenebismethylene linker.

These data support the inhibition of **CXCR4** to prevent the development of **colorectal cancer metastasis**.

The prognostic value of **CXCR4** expression was evaluated by univariate and **multivariate analyses** adjusted by age, sex, Breslow tumor thickness, presence of ulceration, and sentinel **lymph node metastases**.

Logistic regression analysis revealed that positive expression of **CXCR4** protein was an independent and superior predictor for bone **metastasis** to Gleason sum (P < 0.05).

Our data suggest that **CXCR4** might be particularly important in facilitating **metastasis** through the **lymphatic system**.

Using **flow cytometry** in combination with **reverse transcriptase-polymerase chain reaction** (**RT-PCR**), we observed that **bone marrow** CD34(+), CD61(+) cells, **blood platelets**, and **megakaryocytic leukemia cell lines** all expressed the **CXCR4** receptor.

Ovarian cancer cell lines and cells freshly isolated from **ascites** expressed **CXCR4** protein.

Studies using **TAK-779** and **AMD3100** showed that two highly M-tropic isolates entered **microglia** primarily via **CXCR4**.

CXCR4 activation induces epidermal growth factor receptor **transactivation** in an **ovarian cancer cell line**.

Overall survival was significantly correlated with **lymph node metastasis** (952.1 +/- 53.8 d in negative group vs 475.1 +/- 56.2 d in positive group, P = 0.023), distant **metastasis** (874.0 +/- 60.4 d in negative group vs 434.9 +/- 75.2 d in positive group, P = 0.014) and CRT (811.5 +/- 51.2 d in responder group vs 459.6 +/- 94.0 d in non-responder group, P = 0.00038) and further with an absence of **CXCR4** expression or no **residual tumor** (959.8 +/- 51.0 d in null expression or no tumor group vs 412.0 +/- 57.1 d in positive expression group, P = 0.0001).

Expression of anti-sense **CXCR-4** in **glioblastoma cell lines** caused **neurite** outgrowth and cellular differentiation.

We examined roles of **CXCR4** and its ligand, stromal cell-derived factor (SDF)-1, **in migration** of ICC with respect to tumor-stromal interaction by using two ICC **cell lines**, a **fibroblast cell line** (WI-38), and 28 human ICC tissues.

RESULTS: Specific **CXCR4** receptor transcripts were detected in normal **urothelium**, the **bladder cancer cell lines** J82 and T24 and in all bladder carcinoma specimens.

To explore this hypothesis, we phenotyped by fluorescence-activated cell sorter analysis various human **tumor cell lines** for expression of **CXCR4** and found that it was highly expressed on several **rhabdomyosarcoma** (RMS) **cell lines**.

Rapid mobilization of CD34+ cells following administration of the **CXCR4** antagonist **AMD3100** to patients with **multiple myeloma** and **non-Hodgkin's lymphoma**.

In some **cell lines**, **CXCR4** is efficiently modified by a **chondroitin sulfate** chain at **serine** 18, but neither **HIV-1** entry nor stromal derived factor 1 alpha binding was affected by loss of this glycosaminoglycan.

Studies have shown that the HIV envelope protein gp120 binds to neuronal **CXCR4** and activates signal transduction pathways leading to apoptosis.

Our data suggest that **CXCR4** plays an important role in lymphocyte trafficking through tissues, especially between peripheral blood and bone marrow, participating in the regulation of lymphocyte homeostasis in these compartments.

Stromal-derived factor-1 abolishes constitutive apoptosis of WHIM syndrome neutrophils harbouring a truncating **CXCR4** mutation.

Human immunodeficiency virus-induced apoptosis of human hepatocytes via **CXCR4**.

Tumor-cell homing to lymph nodes and bone marrow and **CXCR4** expression in esophageal cancer.

When MOLT4 cells, which expressed Fas as well as **CXCR4**, were stimulated with cycloheximide (CHX), an agonistic anti-Fas antibody, or a combination of these, the cells rapidly underwent apoptosis.

Intracellular **CXCR4** signaling, neuronal apoptosis and neuropathogenic mechanisms of HIV-1-associated dementia.

Kappa-opioid receptor agonist inhibition of HIV-1 envelope glycoprotein-mediated membrane fusion and **CXCR4** expression on CD4(+) lymphocytes.

Down-regulation of surface **CXCR4** was detected in B-lymphoma cells which migrated towards the stromal cells but not in those which showed no migratory response.

Expression of **CXCR4** in eosinophils: functional analyses and cytokine-mediated regulation.

METHODS: Apoptosis occurring in cocultures of chronically HIV-1 IIIB-infected cells with CD4 target cells expressing the **CXCR4** receptor was quantified by terminal deoxinucleotidyl transferase dUTP nick end labeling (TUNEL) or propidium iodide staining followed by fluorescent antibody cell sorting, which allows the evaluation of single-cell killing.

The mutated gene may result in production of the mutant **CXCR4** protein causing abnormal apoptosis and migratory function, which are thought to be related to the cause of chronic neutropenia in WHIM syndrome.

These findings raise the possibility that **CXCR4** function is differentially controlled during B lymphopoiesis and may be relevant to the compartmentalization of B-cell precursors in the bone marrow.

Syncytia arising from the fusion of cells expressing a lymphotropic human immunodeficiency virus (HIV)-1-encoded envelope glycoprotein complex (Env) gene with cells expressing the CD4/**CXCR4** complex undergo apoptosis through a mitochondrion-controlled pathway initiated by the upregulation of Bax.

A small proportion of mesenchymal stem cells strongly expresses functionally active **CXCR4** receptor capable of promoting migration to bone marrow.

Taken together, L-selectin-mediated signals specifically enhance **CXCR4** expression and function, suggesting a novel mechanism for the modulation of lymphocyte activation during cell adhesion and transmigration.

We first showed that uPAR(84-95) stimulated in vitro dose-dependent migration of mouse CD34(+) M1 leukemia cells and inactivated murine CXCR4 [?]. uPAR(84-95) capability to induce mouse HSC/HPC release from bone marrow and migration into the circulation was then investigated in vivo. uPAR(84-95) i.p. administration induced rapid leukocytosis, which was associated with an increase in peripheral blood CD34(+) HSCs/HPCs.

Culturing of monocytes and lymphocytes with lactacystin enhanced the amount of the 101-kDa **CXCR4** isoform in immunoblots by three- to sevenfold.

By immuno-histochemistry, **CXCR4** was detected in the endothelial cells lining sinusoids, arterioles, and venules in the bone marrow.

Incubation of leukocytes with stroma derived factor-1alpha, the natural ligand for CXCR4, induces down-regulation of up to 60% of surface-expressed receptors in a pertussis toxin-insensitive manner.

AMD-3100 is a small molecule inhibitor of HIV-1 attachment to the CXCR4 chemokine receptor, and T-20 is a synthetic peptide corresponding to a region of HIV-1 gp41 that blocks fusion to cell membranes.

Up-regulation of functional CXCR4 expression on human lymphocytes in sepsis.

Chronic lymphocytic leukemia B cells express functional CXCR4 chemokine receptors that mediate spontaneous migration beneath bone marrow stromal cells.

This effect was blocked by the CXCR4 antagonist AMD 3100 (1 microm) but not by the metabotropic glutamate receptor antagonist MCPG (500 microm), indicating a direct action of SDF-1alpha on its cognate receptor.

Compared to the corresponding metastasized tumors in the lymph nodes, primary invasive carcinomas showed more intense staining for CXCR4, particularly on the cellular membrane.

The affinity of AMD3100, a symmetrical nonpeptide antagonist composed of two 1,4,8,11-tetraazacyclotetradecane (cyclam) rings connected through a 1,4-dimethylene(phenylene) linker to the CXCR4 chemokine receptor was increased 7, 36, and 50-fold, respectively, by incorporation of the following: Cu(2+), Zn(2+), or Ni(2+) into the cyclam rings of the compound.

In addition, contact between the lymphoma cells and the stromal cells resulted in down-regulation of surface CXCR4 on the lymphoma cells.

Using methylation-specific PCR, combined bisulfite restriction analysis, and bisulfite sequencing, we found the 5' CpG islands of the CXCR4 gene to be unmethylated in normal pancreas, whereas promoter hypermethylation was detected in 45% (9 of 20) of pancreatic cancer cell lines and in 46% (46 of 100) of primary pancreatic adenocarcinomas.

Transduced signals lead to cell chemotaxis and are terminated through receptor internalization depending on phosphorylation of the C terminus part of CXCR4.

CXCR4 cytoplasmic expression was associated with parameters of tumor aggressivity (tumor grade and lymph node status) and had prognostic value (age-adjusted hazard ratio=1.73; Confidence Interval: 1.07-2.77) with respect to disease-specific survival.

The percentage of nuclear staining increased from normal breast tissue (20%) to ductal carcinoma-in-situ DCIS (43%) to invasive cancer (67%) while CXCR4 was expressed in the cytoplasm of 67% of (DCIS) cases (double that in normal breast samples), suggesting an important role in breast tumor progression.

Notably, CXCR4 was significantly over-expressed in cancerous lesions (carcinomas and metastasis) compared to non-cancerous lesions (normal mucosa and polyps) (P = 0.003) and in adenomatous polyps versus hyperplastic polyps (P = 0.009).

The expression of functional CXCR4 on the cell surface was demonstrated by the detection of ligand-induced Ca(2+) mobilization, chemotaxis, and ligand-induced receptor endocytosis.

SUMMARY: CXCR4 participates in several biological processes (bone marrow hematopoiesis, cardiogenesis, angiogenesis, neurogenesis) and is implicated in different clinical pathologic conditions (WHIM, HIV infection, tumor metastatization, autoimmunity).

Treatment with CXCR4 antagonists resensitized CLL cells cultured with stromal cells to fludarabine-induced apoptosis.

Immunohistochemistry revealed overexpression of both CXCR4 and SDF-1alpha within tumor cells and endothelial cells of hemangioblastomas and CC-RCCs.

The most potent interaction with CXCR4 and thus anti-HIV activity was shown by bicyclam analogs with cyclam rings composed of fourteen members that are linked by an aromatic (phenyl) bridge.

Glucose transporter 1 expression identifies a population of cycling CD4+ CD8+ human thymocytes with high CXCR4-induced chemotaxis.

The functional significance of CXCR4 expression on cells of the megakaryocytic lineage was examined by studying the effects of SDF-1alpha on migration and proliferation of megakaryocyte progenitor cells in vitro.

As well as being important for lymphocyte trafficking and recruitment at sites of inflammation, it appears that CXCR4 and its ligand stromal cell-derived factor-1 play an important role in hematopoiesis and developmental processes such as organogenesis, vascularization and embryogenesis.

The 73-kDa heat shock cognate protein is a CXCR4 binding protein that regulates the receptor endocytosis and the receptor-mediated chemotaxis.

Therefore, CXCR4 activation of eosinophils seems to be important in the chronic phase of allergic reaction, which is dominated by a Th1 cytokine profile.

Here we show that primary intestinal (jejunal) epithelial cells express galactosylceramide, an alternative primary receptor for HIV-1, and CCR5 but not CXCR4.

In contrast, the differentiation of CXCR4- B cells into plasma cells is generally accompanied by the induction of CXCR4 expression.

Additionally, semen-derived viral populations exhibited constrained diversity ($P < 0.05$), decreased levels of positive selection ($P < 0.025$), decreased CXCR4 coreceptor utilization, and altered glycosylation patterns.

We used the inducible HL-60 cell line as a model system for comparative analysis of CXCR4 expression during differential maturation into the granulocytic or monocytic phenotypes.

Stromal cell-derived factor-1 from biliary epithelial cells recruits CXCR4-positive cells: implications for inflammatory liver diseases.

The inefficient, CXCR4-mediated infection of differentiated HCT116 cells supports the view that epithelial cells are a barrier and not a portal for HIV transmission.

Prostaglandin-induced human endothelial cell organization and subsequent vascularization can be inhibited to a greater extent by a neutralizing antibody to human CXCR4 in severe combined immunodeficient mice.

Differential expression of stromal cell-derived factor 1 and its receptor CXCR4 in the skin and endothelial cells of systemic sclerosis patients: Pathogenetic implications.

Flow cytometric analysis revealed that this effect may be due largely to up-regulation of CXCR4 expression by SDF-1alpha on CrFK cells, an effect mimicked by treatment of the cells with phorbol myristate acetate.

SDF-1alpha activates basophils to chemotaxis (chemotactic index = 3.8) and histamine release (36% of total content) through CXCR4 on the cells.

The alpha-glucosidase inhibitor 1-deoxynojirimycin blocks human immunodeficiency virus envelope glycoprotein-mediated membrane fusion at the CXCR4 binding step.

Exposure of ACC cells to cisplatin resulted in upregulation of CXCR4 on the cell surface, which was repressed by the transcriptional inhibitor, alpha-amanitin.

We maintained expanded BM- or CB-derived MSCs for up to 15-18 passages with monitoring of the expression of 1) various tissue markers (cardiac and skeletal muscle, neural, liver, and endothelial cells), 2) functional CXCR4 and c-met, and 3) MMPs.

RESULTS: RU24858 (1 muM) increased CXCR4 and Annexin 1 expression on eosinophils to a similar extent as mometasone (1 muM) and dexamethasone (1 muM).

Laminar shear stress inhibits **CXCR4** expression on **endothelial cells**: functional consequences for **atherogenesis**.

Impaired **CXCR4** signaling contributes to the reduced neovascularization capacity of endothelial **progenitor cells** from patients with **coronary artery** disease.

We found that **CXCR4** phosphorylated on **serine** 339 was present in tumor cells and **vascular endothelial cells** in all grades of **astrocytoma**.

Mutation of the three **lysine** residues had no effect on **CXCR4** **endocytosis** yet completely inhibited receptor degradation.

Therefore, our data strongly suggest that the **CXCR4** receptor-mediated intracellular signaling pathway of gp120 differs in **astrocytes** and neurons.

The recombinant receptor undergoes N-linked **glycosylation**, **tyrosine** sulfation and is recognized by the 12G5 conformation specific antibody against human **CXCR4**.

Surprisingly, when placed in a myeloid culture environment, the **CXCR4**(-) **B-cell** progenitors could differentiate into **granulocyte**, macrophage, and **erythroid cells** at a high frequency.

CXCR4 may influence **cell migration** in the **peritoneum**, a major route for **ovarian cancer** spread, and could be a therapeutic target.

The aim of this study was to assess the anti-CXCL12/**CXCR4** activity of a commercial **tannic acid** and evaluate its potential to inhibit tumor **cell migration** and angiogenesis **in vitro**.

We now demonstrate that **DNA methylation** influences **CXCR4** expression in human **pancreatic cancer**.

Regulation of **cellular proliferation**, cytoskeletal function, and **signal transduction** through **CXCR4** and c-Kit in **small cell lung cancer** cells.

Here we report that the **human immunodeficiency virus** co-receptor **CXCR4** undergoes rapid agonist-promoted degradation by a process involving **endocytosis** via clathrin-coated pits and subsequent sorting to **lysosomes**.

The small number of mutations in viruses selected for **CXCR4** use were distinctly nonrandom, with a dominance of charged **amino acid substitutions** encoded by G-to-A transitions, changes in N-linked **glycosylation** sites, and isolate-specific mutation patterns.

In response to ligand stimulation, **CXCR4** underwent internalization and colocalization with Hsc73, but the receptor **endocytosis** was blocked by knockdown of Hsc73 with **RNA interference**.

We have therefore investigated whether these nucleotide **polymorphisms** will reduce the expression levels of surface CCR5 and **CXCR4**, and thus lead to slower AIDS progression.

We conclude that alpha4beta1 **integrin** binding and **CXCR4** chemokine receptor activation are prerequisites for the migration of CD34+ haematopoietic progenitors and AML cells beneath marrow **stromal cells**.

Agonist-independent **endocytosis** of **CXCR4** occurs through **clathrin-coated vesicles**.

Because SDF-1alpha and **CXCR-4** are felt to be involved in **progenitor cell** homing to marrow, the abnormality decribed here could contribute to the homing and retention defects typical of immature **myeloid cells** in **chronic myelogenous leukemia**.

The **CXCR4** ligand **stromal-cell** derived factor-1 induced Ca2+ fluxes and inhibited both constitutive and **vitamin D3**-enhanced **HIV** replication in minus clones.

The NMR structures of the d-peptides provide a structural basis to understand their mechanism of action and design new peptidomimetic analogs to further explore the **structure-activity relationship** of d-peptide ligand binding to **CXCR4**.

A comparison of wild-type (WT) and dual N-linked **glycosylation site**, N11A/N176A, mutant **CXCR4** expressed in 3T3 and HEK-293 cells

served to implicate variabilities in **glycosylation** and oligomerization in almost half of the isoforms.

We studied the distribution of stromal cell-derived factor 1 isoforms alpha and beta, and their receptor **CXCR4**, in **polymyositis**, **sporadic inclusion body myositis** and **dermatomyositis** using **in situ hybridization**, **immunohistochemistry**, **immunofluorescence** and **Western blotting**.

Reduction of **CXCR4** **energy transfer** by the TM4 peptide and **methyl**-beta-cyclodextrin indicates that interactions between CXCR4s may play important roles in **cell migration** and suggests that cell surface and intracellular receptor dimers are appropriate targets for control of tumor cell spread.

Conversely, **CXCR4** was expressed by **chondrocytes** but not by synovial **fibroblasts**.

The **sulfate** group at **tyrosine** 21 contributes substantially to the ability of **CXCR4** to bind its ligand, stromal derived factor 1 alpha.

Multivariate analysis revealed that mRNA expression level of **CXCR4** was the only significant variable for overall survival (P = 0.0006), **event-free survival** (P = 0.004), and MFS (P = 0.025).

The presence of **CXCR4** on the surface of cultured but not freshly isolated **Langerhans cells** has been described.

CXCR4 modulates contractility in adult **cardiac myocytes**.

Flow cytometry and immunostaining studies were performed, which demonstrated that the **HIV-1** receptors CD4, CCR5 and **CXCR4** were not expressed by villous **trophoblast** cells.

Cross-sectional analysis of human immunodeficiency virus-exposed, uninfected infants revealed high proportions of **CXCR4**-expressing cells in their **cord blood**, which declined at 4.5 months and increased between 9 and 15 months to levels approaching those of uninfected adults.

Exclusion of **CXCR4** as the cause of trapped **neutrophil** syndrome in Border Collies using five **microsatellites** on canine **chromosome** 19.

Furthermore, activation of the **SDF-1 receptor** CXCR4 stimulated BG-1 and MCF-7 **cell proliferation** in a manner comparable to **estradiol**.

Transfection of an expression vector containing the **CXCR4** c-DNA rendered UT-7 cells readily infectable by different T-lymphotropic syncytium-inducing **HIV-1** and **HIV-2** isolates.

The physical association of **CXCR4** and CD26, direct or part of a supramolecular structure, suggests a role on the function of the **immune system** and the pathophysiology of **HIV** infection.

The coreceptor activity of the second extracellular loop of **CXCR4**, which is restricted to dual tropic and T-tropic strains, was insensitive to the removal of charged residues either singly or in combinations by **alanine** scanning **mutagenesis** or to the conversion of acidic residues to **lysine**.

Tumor necrosis factor (TNF)-alpha pretreatment of ICC cells up-regulated **CXCR4** mRNA and protein expression in a concentration-dependent manner.

It was demonstrated that human **mast cells** constitutively express mRNA and protein for **CXCR4**.

For this study, by employing **alanine**-scanning **mutagenesis**, (125)I-SDF-1alpha competition binding, **Ca(2+)** mobilization, and cell-cell fusion assays, we found that the mutation of many **CXCR4** TM residues, including Tyr(45), His(79), Asp(97), Pro(163), Trp(252), Tyr(255), Asp (262), Glu(288), His(294), and Asn(298), could selectively decrease HIV-1-mediated **cell fusion** but not the binding activity of SDF-1alpha.

We introduce a simple bioinformatic method of scoring V3 **amino acid sequences** that reliably predicts **CXCR4** usage (sensitivity, 84%; specificity, 96%).

Th2 polarized cells expressed more **CXCR4** than **Th1 cells**.

AMD3465, a monomacrocyclic CXCR4 antagonist and potent HIV entry inhibitor.

HIV-1 infection of frontal lobe microglia was 100% successful using both CXCR4- and CCR5-tropic strains of HIV-1.

The vast majority of inflammatory cells in idiopathic inflammatory myopathies were CXCR4 positive.

Full-length genome analysis of HIV-1 subtype C utilizing CXCR4 and intersubtype recombinants isolated in South Africa.

CXCR4 gene homologues were isolated from an ape (gibbon), an Old World monkey (African green monkey), and two New World monkeys (squirrel monkey and cotton-top marmoset), and their DNA sequences determined.

Studies on CXCR4 expression and regulation in neuroepithelial cells are fundamental for understanding its physiopathologic roles in the central nervous system (CNS).

CXC chemokine receptor 4 (CXCR4) is a co-receptor for human immunodeficiency virus (HIV) infection and is believed to be involved in the pathogenesis of AIDS-associated neurologic disorders and brain tumors.

About 70% CXCR4 was reduced by ampelopsin at 1 mg/mL.

CXCR4 expression is a prognostic marker in various types of cancer, such as acute myelogenous leukemia or breast carcinoma.

Phenotypic and functional evidence for the expression of CXCR4 receptor during megakaryocytopoiesis.

As occurs with the cytokine receptors in response to cytokines, the CXCR4 undergoes receptor dimerization after SDF-1alpha binding and is a critical step in triggering biological responses.

Although CXCR4 [?] blockade selectively targeted endothelium generated by vasculogenesis, completely inhibiting vessel formation may require combination therapy targeting locally derived and marrow-derived endothelium.

We suggest that non-peptide antagonists with, for example, improved oral bioavailability can be designed to mimic this interaction and thereby efficiently and selectively block the CXCR4 receptor.

The proteasome inhibitors also inhibited the SDF-1alpha and gp120 protein-induced down-modulation of the CXCR4 receptor in Jurkat cells.

Conversion of Asn-119 to Ser or Ala, but not Asp or Lys, conferred autonomous CXCR4 signaling in yeast and mammalian cells.

Experiments with actinomycin D demonstrated that CXCR4 transcripts were short-lived, indicating a rapid mRNA turnover.

Cloning and analysis of the promoter region of CXCR4, a coreceptor for HIV-1 entry.

Unique ligand binding sites on CXCR4 probed by a chemical biology approach: implications for the design of selective human immunodeficiency virus type 1 inhibitors.

AMD2763, 1,1'-propylene-bis(1,4,8, 11-tetraazacyclotetradecane), which is a less potent CXCR4 [?] antagonist, was virtually inactive against FIV in feline thymocytes (IC50, >66.5 microgram/ml), while it was clearly active in CRFK cells (IC50, 0.9 microgram/ml).

This X4-dependent restriction of HIV replication was not explained by either the absence of functional CXCR4 on the cell surface or by the inefficient viral entry and reverse transcription.

Although early and persistent SI variants have been described in longitudinal studies, this is the first demonstration of exclusive and persistent CXCR4 usage.

Allergen inhalation attenuated both intensity of CXCR4 expression and SDF-1alpha levels in marrow from dual compared with early responders 24 hours postallergen.

We conclude that the native form of cholesterol with the hydroxyl group at C3 is critical to CXCR4 and CCR5 conformation and function.

Subtype-specific expression and genetic alterations of the chemokinereceptor gene **CXCR4** in **medulloblastomas**.

In contrast, **alanine** or **phenylalanine** substitution at Cys-11 led to significant enhancement in peptide affinity for **CXCR4**.

Taken together, these results support the possibility that the neuroactive effects of FGF in **HIV encephalitis** might be mediated through regulation of the expression of **CXCR4**.

In 66% of the samples, atypical ductal **hyperplasia** was present, and > 92% exhibited positive **CXCR4**-staining.

CXCR4 is expressed in **ductal carcinoma** in situ of the breast and in atypical ductal **hyperplasia**.

Modulation of neuronal **CXCR4** by the mu-opioid agonist **DAMGO**.

The physiological roles of **CXCR4** in developmental patterning of the nervous and **hematopoietic system**; gastrointestinal angiogenesis; and cardiac **organogenesis** were established by studies in gene-targeted mice.

There was no difference in the **CXCR4** mRNA expression in colon, esophageal or **gastric cancers** compared to non-cancerous tissues.

CXCR4 mRNA expression in colon, esophageal and **gastric cancers** and **hepatitis C** infected liver.

Tumor necrosis factor-alpha drives **HIV-1** replication in **U937 cell** clones and upregulates **CXCR4**.

Several peptidic compounds, T22 (an 18-mer), T134 (a 14-mer), ALX40-4C (a 9-mer) and **CGP 64222** (also a 9-mer), have been identified as **CXCR4** antagonists and show anti-HIV activity.

This increase in **CXCR4** expression level was inhibited by the addition of **brefeldin A**, **actinomycin D**, or **cycloheximide**.

Surprisingly, the **CXCR4** promoter (nucleotides -1098 to +59) fused to luciferase was found to be activated similarly in CEM and **Jurkat cells** in response to **DcAMP** in a concentration-dependent manner.

The **limbic system** plays a key role in memory, and the presence of **CXCR4**-which can bind the viral envelope protein gp120-min a subset of neurons from this system may play a role in the development of HIV-related **dementia**.

The functional significance of **CXCR-4** expression was ascertained by assessing the promoting effect of SDF-1alpha on **cell cycle**, proliferation, and colony formation.

This segment was designated as a **LOH** cluster region 1 (**LCR 1**).

We have stably expressed **CXCR-4** on mink lung Mv-1-lu and feline kidney **CCC** cells (normally restrictive to **HIV** entry) and have shown efficient fusion, entry, and replication of ROD/B.

Genetic manipulations have revealed new regulatory aspects, including the role of Six transcription factors and the **CXCR4** cytokine receptor during embryonic **myogenesis**.

CXCR4 was internalized through coated pits and **coated vesicles** and subsequently localized in endosomal compartments from where it could recycle to the cell surface after removal of the phorbol ester.

We show that there is a pocket in a model of the human **CXCR4** coreceptor in which trans and cis configurations of metallobicyclam can bind by direct metal coordination to carboxylate side chains, cyclam-NH...carboxylate H bonding, together with **hydrophobic interactions** with **tryptophan** residues.

KRH-1636 prevented monoclonal antibodies from binding to **CXCR4** without down-modulation of the coreceptor.

Result page: **1** 2 **[Next]**

Symbol	Name	Synonyms	Organism
CXCL12	chemokine (C-X-C motif) ligand 12 (stromal cell-derived factor 1)	chemokine, hIRH, PBSF, Pre-B cell growth-stimulating factor, SCYB12, SDF1, SDF-1, SDF1A, SDF-1a, SDF1B, SDF-1b, Stromal cell-derived factor 1 precursor, TLSF-a, TLSF-b, TPAR1	Homo sapiens

UniProt	P48061, Q8NF00, Q6ICW0
PDB Structure	1QG7, 1A15
OMIM	609423, 600835
NCBI Gene	6387
NCBI RefSeq	NP_000600, NP_954637
NCBI RefSeq	NM_199168, NM_000609
NCBI UniGene	6387
NCBI Accession	AI092156, AAB39333

Homologues of CXCL12 ...

Interaction information for this gene ...

Breaking news for this gene ... new

Enhanced PubMed/Google query ...

However, the chemotactic response of both polymorphonuclear cells and T lymphocytes in response to **CXCL12** is increased.

The **chemokine** RANTES (regulated on activation, normal T cell expressed and secreted) is a potent regulator of leukocyte trafficking.

The pattern of expression of these **chemokine** receptors on T cell subsets and their regulation has important implications for AIDS pathogenesis and lymphocyte recirculation.

Stromal cell- derived factor (SDF)-1alpha is a CXC **chemokine** previously characterized as an efficacious chemoattractant for T lymphocytes and monocytes in peripheral blood.

Interleukin (IL)-8, a C-X-C **chemokine**, is a potent chemoattractant and an activator for neutrophils, T cells, and other immune cells.

Cutting edge: C-C **chemokine** receptor 6 is essential for arrest of a subset of memory T cells on activated dermal microvascular endothelial cells under physiologic flow conditions in vitro.

These data demonstrate that colonic epithelial cell **chemokine** production can be differentially regulated by T cell-derived cytokines and suggest an interplay between epithelial cells and T lymphocytes potentially important in the intestinal inflammation.

Similarly, CP55,940 and JWH-015 inhibited the **CXCL12**-induced chemotaxis of primary CD4(+) and CD8(+) T lymphocytes.

Persistent alterations in T-cell repertoire, cytokine and **chemokine** receptor gene expression after 1 year of highly active antiretroviral therapy.

Of these, the **chemokine** RANTES is responsible for the recruitment of inflammatory cells such as eosinophils and T-lymphocytes.

CC **chemokine** ligand 19 secreted by mature dendritic cells increases naive T cell scanning behavior and their response to rare cognate antigen.

The role of cell surface proteoglycans in CC **chemokine**-mediated anti-HIV-1 activity in T cells and **macrophages** was investigated.

The role of the C-C **chemokine** receptor-5 Delta32 **polymorphism** in asthma and in the production of regulated on activation, normal T cells expressed and secreted.

CXC **chemokine** receptor 5 expression defines follicular homing T cells with **B cell** helper function.

These results indicate that activation of Vav1-Rac signaling pathway by **CXCL12** represents an important inside-out event controlling efficient **up-regulation** of alpha4beta1-dependent T lymphocyte adhesion.

SDF-1 stimulated migration of rheumatoid synovial T cells and also inhibited activation-induced **apoptosis** of T cells.

The proliferation history and T cell receptor repertoire of these **chemokine**-receptor(+) cells suggest that they are very early memory CD4(+) T cells that have "rested down" before acquiring the **phenotypes** described for "central" or "effector" memory T cells.

The role of **chemokine**-matrix interactions in **integrin**-dependent T-cell migration was examined to address the critical question of how chemokines provide directional information.

Our results suggest that human **mast cells** may be a source of multiple chemokines, that glucocorticoids may inhibit the expression of only a subset of these chemokines, and that **mast cells** and T-cell **chemokine** expression may occur via distinct regulatory pathways.

Together, these findings support a pivotal role for HEC-expressed **CXCL12 [?]** and its receptor on T cells in the regulation of T lymphocyte homing to **lymph nodes**.

However, the enhanced **chemokine**-induced migration by memory T cells across activated **endothelium** appears to be independent of the increase in endothelial CAM expression.

To explore potential mechanisms, we evaluated the **signal transduction** pathways activated by **SDF-1** in the Jurkat T cell line.

Expression of **chemokine** receptors on activated T cells is important in allowing these cells to traffic into and accumulate within the **central nervous system** (CNS) of MHV-infected mice.

The patterns of **chemokine** receptor expression suggest that X4 and R5 viruses have a preferential **tropism** for naïve and memory T cells, respectively.

To investigate the ability of RXM to downregulate skin-infiltration of T-lymphocytes, we examined the effects of RXM on **keratinocyte** production of chemokines and T cell expression of **chemokine** receptors.

Th1 and **Th2 cells** are functionally distinct subsets of CD4+ T lymphocytes whose tissue-specific homing to sites of inflammation is regulated in part by the differential expression of P- and E-selectin ligands and selected **chemokine** receptors.

Response to treatment and **disease progression** linked to CD4+ T cell surface CC **chemokine** receptor 5 density in **human immunodeficiency virus** type 1 vertical infection.

Immunohistochemistry shows significant infiltration of **SDF-1 [?]** tumors by T cells, and in vivo T-cell depletion studies indicate that CD4(+) T cells are required for SDF-mediated tumor rejection.

Chemokine receptor expression on T cells is related to new lesion development in **multiple sclerosis**.

This suggests that T-cell recruitment to human **lymphoid tissues** depends on the transcytosis of lymphoid chemokines through HEV cells because there is at present no evidence of alternative **chemokine** production in these cells that could explain the attraction of naive T lymphocytes.

METHODS: Expression of activation markers and **chemokine** receptors from blood and **bronchoalveolar lavage** (BAL) fluid T cells and the T(H)2 cytokine expression from these T cells and bronchial **mucosa** biopsy specimens were assessed from subjects with eosinophilic

bronchitis, subjects with asthma, and healthy control subjects.

Chemokine Receptor Expression in Cutaneous T cell and NK/T-cell Lymphomas: Immunohistochemical Staining and In Vitro Chemotactic Assay.

Chemokine receptors expressed on T cells in packed red blood cell units change over storage time.

Here, we report that intratumoral expression of the **chemokine** CCL21 enhances the efficacy of adoptive T-cell therapy in a mouse model of melanoma.

The present study shows that a CC **chemokine** CCL19 attracts mature T cells out of the fetal thymus organ culture.

SDF-1 is also a potent chemoattractant for T cells and has roles in both inflammation and immune homeostasis.

Differential expression of binding sites for **chemokine** RANTES on human T lymphocytes.

Considering the T-cell-attracting and -stimulating capacity of Mig and the importance of T-cells in the pathogenesis of psoriasis, this study indicates that this novel C-X-C **chemokine** plays an important role as a mediator of T-cell recruitment and activation in the papillae and thus contributes significantly to the cytokine network of inflammation in psoriasis.

Increased adhesion molecule and **chemokine** receptor expression on CD8+ T cells trafficking to cerebrospinal fluid in HIV-1 infection.

Conclusion Allergen-specific induction of cytokines and chemokines in PBMC and **chemokine** receptor expression on circulating T cells may contribute to the pathogenesis of NRL allergy.

Chemokine receptor expressions and responsiveness of cord blood T cells.

We report that Cbl family members, Cbl and Cbl-b, are tyrosine-phosphorylated after SDF-1alpha/**CXCL12** stimulation of Jurkat T cells.

T cells initially formed an antigen-independent 'tethered' adhesion on **chemokine**-bearing antigen-presenting cells.

Endothelial induction of the T-cell **chemokine** CCL21 in T-cell autoimmune diseases.

These cytokines generated expression of the **chemokine** CXCL9 in the liver, thereby enhancing CD8+ T cell infiltration and liver disease in mice.

These data suggest that the **SDF-1**-mediated cell survival combined with its priming function would set T cells to respond to immunologic challenges.

Here, we detected increasing secretion of the **chemokine** RANTES (regulated upon activation, normal T cell expressed and secreted), which functions to recruit the immune cells, in dengue-virus-infected liver cells and patients.

We show that newly identified **chemokine** receptor Bonzo/CXCR6 is expressed by subsets of Th1 or T-cytotoxic 1 (Tc1) cells, but not by Th2 or Tc2 cells, establishing Bonzo as a differential marker of polarized type 1 T cells in vitro and in vivo.

Using double-chamber cultures and activated T cell plasma membrane preparations we demonstrated that both cell contact and soluble factors contributed to RTEC **chemokine** production.

Granulosa cells from follicular aspirates produce **CXCL12** that contributes to T lymphocytes recruitment.

In vivo, antigen-induced T-cell recruitment into the peritoneal cavity was reversed by high but not low concentrations of **SDF-1**.

Chemokine stimulation of human peripheral blood T lymphocytes induces rapid dephosphorylation of ERM proteins, which facilitates loss of microvilli and polarization.

The CXC **chemokine** stromal cell-derived factor activates a Gi-coupled phosphoinositide 3-kinase in T lymphocytes.

Estradiol and interleukin-1beta exert a synergistic stimulatory effect on the expression of the **chemokine** regulated upon activation, normal T

cell expressed, and secreted in endometriotic cells.

Chemokine receptor expression on neoplastic and reactive T cells in the skin at different stages of **mycosis fungoides**.

AMD-3100, a **bicyclam**, is a novel agent that uniquely inhibits the entry of **human immunodeficiency virus** type 1 (**HIV-1**) into CD4(+) T cells via selective blockade of the **chemokine** CXCR-4 receptor.

Abnormal expression of intracellular cytokines and **chemokine** receptors in peripheral blood T lymphocytes from patients with **systemic sclerosis**.

Activation with mycobacterium-derived, phosphate-containing components, modulated the **chemokine** receptor profile of gammadelta T lymphocytes as well as their pattern of cyto-chemokine production, disclosing a potential for their active participation in **granuloma** formation.

FTY720 exerts differential effects on CD4+ and CD8+ T-lymphocyte subpopulations expressing **chemokine** and adhesion receptors.

Higher levels of activation markers and **chemokine** receptors on T lymphocytes in the **cervix** than peripheral blood of normal healthy women.

Cortisol or postexercise plasma treatment markedly enhanced migration of T lymphocytes toward **CXCL12**.

Natural killer T cells infiltrate **neuroblastomas** expressing the **chemokine** CCL2.

The pattern of **chemokine** receptor expression in T-cell **non-Hodgkin lymphoma** has not been previously studied.

These data demonstrate that **histamine** activates immature DC and induces **chemokine** production, thereby suggesting that **histamine**, via stimulation of resident DC, may participate locally in T cell stimulation and in the late inflammatory reaction associated with allergic disorders.

We initially sought to identify which chemokines were produced by a range of human **tumor cell lines**, and which chemokines and **chemokine** receptors were expressed by cultured T cells.

METHODS: For a more detailed comprehension of the pathogenesis of T-cell recruitment in human acute rejection, the in situ expression of chemokines and **chemokine** receptors in **allografts** of 26 patients between day 3 and 9 after **renal transplantation** was examined in the present **prospective study**.

OBJECTIVE: In order to assess the contribution of these molecules to the local recruitment of T cells in **bronchial asthma**, we analysed the expression of 14 **chemokine** receptors on lung-derived T cells.

Using the human T-cell line Jurkat, we have confirmed previous observations that pre-incubation with **met-enkephalin** (MetEnk), an endogenous opioid agonist, prevents the subsequent chemotactic response to the **chemokine** RANTES.

Direct and indirect effects of **retinoic acid** on human Th2 cytokine and **chemokine** expression by human T lymphocytes.

Calcium ion mobilization, an important first step in **chemokine** receptor signaling, was subsequently demonstrated in transduced T cells in response to Gro-alpha.

In addition, the **Reed-Sternberg cells** produce the **chemokine** TARC that could lead to the specific attraction of a Th2 T-cell subset.

The ability to use whole blood culture to estimate **chemokine** expression in T cell subsets may ultimately provide a practical means to evaluate disease status and to monitor **early intervention** therapies which target chemokines.

CONCLUSIONS: **Selective** expansion of CD4+, CD45RO+ memory-type T cells could reflect an antigen-specific and/or **chemokine**-mediated effect in HAG.

CONCLUSION: Increase in secretion of Th1 and Th2 cytokines together with induced expression of **chemokine** receptors on T cells and **monocytes** suggest restoration of peripheral **cell mediated immunity** and blockade of the accumulation of inflammatory cells in joints as response to treatment.

The **chemokine** RANTES has the potential to influence the migration of memory T cells and monocytes across the **blood-retinal barrier** during inflammatory eye disease.

Distinct T cell/renal tubular **epithelial cell** interactions define differential **chemokine** production: implications for tubulointerstitial injury in chronic **glomerulonephritides**.

T cell **chemotaxis** and **chemokine** release after **Staphylococcus aureus** interaction with polarized airway **epithelium**.

Here we show that **B lymphocytes**, **NK cells** and, to a lesser extent, **T lymphocytes** inactivate **SDF-1** by N-terminal processing.

Similarly, **damnacanthal** was shown to inhibit **CXCL12**-induced **chemotaxis** of the Jurkat **T-cell** line.

Gene expression profiles in patients with **lung inflammation** showed increased expression of chemokines and **chemokine** receptor genes, which would lead to migration of **T cells**, especially type 2 **T cells**, and phagocytic cells.

Tissue distribution analysis demonstrates that **dendritic cells** present in germinal centres and **T-cell** areas of secondary lymphoid organs express this **chemokine**.

Adenosine affects expression of membrane molecules, cytokine and **chemokine** release, and the **T-cell** stimulatory capacity of human **dendritic cells**.

Together, these findings support a model of CD8(+) **T cell** memory **cell differentiation** involving the delivery of key signals early in the priming process based on **chemokine**-guided attraction of naive CD8(+) **T cells** to sites of Ag-driven interactions between TLR-activated **dendritic cells** and CD4(+) **T cells**.

We first studied the effects of various physiologic concentrations of **progesterone** on the expression of chemokines and **chemokine** receptors by **T cells** and **macrophages**.

To address this question, this study was carried out in order to determine the frequencies of the **SDF1** **polymorphism** and the SDF1-3'A allele on 1061 genomic DNA samples purified from peripheral **blood cells** of 136 healthy individuals (group 1), 147 HIV-1-exposed seronegative individuals (group 2), 161 HIV-1-infected asymptomatic individuals and with CD4(+) **T-cells** count 350 mm(-3) (group 3), and 617 HIV-1-infected individuals with AIDS and/or CD4(+) **T-cells** count < 350 mm(-3) (group 4).

We examined the expression of **chemokine** receptors on the surfaces of **T cells** and **B cells** from 27 individuals either with lymphatic filarial disease (**lymphedema**), with the asymptomatic or subclinical form of filarial infection, or without filarial infection.

Statins inhibit the inducible expression of **major histocompatibility complex** class II in several cell types including **macrophages** and downregulate the expression of T-helper-1 (Th1) **chemokine** receptors on **T cells**, leading further to inhibition of activation of **lymphocytes** and their infiltration into the inflammation sites.

Moreover, cellular response **kinetics** appeared to further correlate with the **up-regulation** of endogenous **T cells** producing the **chemokine** IFN-gamma-inducible protein-10 in vivo.

Finally, no distinct differences in **chemokine** production were observed when the responses to **T cell** contact or to prototypic Th1 and Th2 cytokines were examined in **systemic sclerosis** versus normal **fibroblasts**.

Chemokine receptors on **T cells** are frequently categorized as functioning either in **immune system** **homeostasis** within lymphoid organs, or in peripheral inflammation.

Analysis of **NK cells** and **chemokine** receptors in tumor infiltrating CD4 **T lymphocytes** in human renal **carcinomas**.

In support of this, we show that liver-infiltrating **lymphocytes** in PSC include mucosal **T cells** recruited to the liver by aberrant expression of the gut-specific **chemokine** CCL25 that activates alpha4beta7 binding to mucosal addressin **cell adhesion** molecule 1 on the hepatic **endothelium**.

To determine which **chemokine [?]** receptors might be involved in **T lymphocyte** localization to the **intestinal mucosa**, we examined receptor expression on human intestinal **lamina propria lymphocytes** (LPL), intraepithelial **lymphocytes** (IEL) and CD45RO+beta7hi gut homing peripheral blood **lymphocytes** (PBL).

It is concluded that **calcitriol** has a strong antiproliferative activity and does not interfere with KC responsiveness to gamma-IFN and IL-alpha induced **chemokine** expression or with the adhesion of **T-cells** to **keratinocytes**.

This **chemokine** may be an important attractant and activator of **macrophages**, **T lymphocytes** and/or **eosinophils** in the **uterus** during the reproductive cycle or implantation.

We have determined the structure of the **chemokine** RANTES (regulated on activation normal **T cell** expressed) in the presence of **heparin**-derived **disaccharide** analogs by **X-ray crystallography**.

We thoroughly investigated, ex vivo and **in vitro**, the regulation of **chemokine** receptor expression on human FOXP3(+) **T cells** in neonatal **cord blood**, adult peripheral blood, and **tonsils**.

The aim of this study was to characterize **chemokine** receptor expression in peripheral blood memory **T cells** in **Crohn's disease** (CD) and **ulcerative colitis** (UC), and to correlate the expression with disease activity.

OBJECTIVE: To define the relationships between levels of **chemokine** receptor (CCR)5+ **T-cells** in blood and **cerebrospinal fluid** (CSF) of **optic neuritis** (ON) and control patients (CON).

Membrane rafts also seem to be involved in many other aspects of **T cell** biology, such as functioning of cytokine and **chemokine** receptors, adhesion molecules, **antigen presentation**, establishing **cell polarity** or interaction with important pathogens.

Herein, we report that these compounds effectively inhibited **SDF-1 [?]**-induced migration of human **breast cancer** cells (MDA-MB-231), human leukemia **T cells** (Sup-T1) and human **umbilical vein endothelial cells** at concentrations of 10-100 nM **in vitro**.

We examined the role of nonreceptor **tyrosine** kinases in **CXCL12**-induced **chemotaxis** of **T cells** and natural killer **(NK) cells**.

Based on our results, we suggest that the altered **leukocyte** response to **CXCL12** may account for the pathologic retention of mature polymorphonuclear cells in the **bone marrow** (myelokathexis) and for an altered **lymphocyte** trafficking, which may cause the **immunophenotyping** abnormalities observed in WHIM patients.

The **chemokine** STCP-1 does not induce Ca2+ mobilization in **monocytes**, **dendritic cells**, **neutrophils**, **eosinophils**, **lipopolysaccharide**-activated **B lymphocytes**, and freshly isolated resting **T lymphocytes**.

Astrocyte chemokine expression might contribute to site-specific **leukocyte** infiltration within the CNS.

In addition, analysis of the regulation of **integrin** function and **chemokine**-mediated migration has highlighted the critical role that spatial localization of signaling molecules plays in **signal transduction**, and the importance of the actin **cytoskeleton** in **T cell** function.

The aim of this study was to investigate the effect of the **arginine**-specific **cysteine** protease gingipain-R produced by P. gingivalis on **chemokine** production by human gingival **fibroblasts** (HGF) and the effect of gingipain-R treatment on the subsequent contact-dependent activation of HGF by **T cells**.

Using monoclonal antibodies classified by the Cytokine/**Chemokine** section of the 8th International Workshop on Human **Leukocyte** Differentiation Antigens, we analyzed human **lymphocytes** in blood samples drawn from the **umbilical cord**, normal adults, allergic and non-allergic asthma patients, **HIV** infected, and AIDS positive subjects.

Among 30 viral isolates obtained from peripheral blood, **tropisms** for both human blood-derived cells (**macrophages**, **T-lymphocytes**), and for human neural (brain-derived) cells (**microglia**, **astrocytes**) were determined, as was **chemokine** co-receptor usage.

Monocyte phagocytosis of pathogens or inflammatory debris leads to **chemokine** secretion and heralds the influx of **leukocytes** to the site

of injury.

These constructs were employed in cell fusion and virus infectivity assays using peripheral blood mononuclear cells, MT4 T cells, primary monocyte-derived macrophages, or HOS-CD4 cell lines, expressing various chemokine receptors, to assess the contributions of different gp120 subdomains in coreceptor usage and cellular tropism.

To evaluate the role of H. pylori lipopolysaccharides (LPS) in the recruitment of leukocytes to the gastric mucosa, we have examined the cytokine and chemokine production from human monocytes stimulated with LPS isolated from different H. pylori strains, as well as from several other gram-negative bacteria.

It has been suggested that heparan sulfate on cell surfaces could provide specific ligand sites on endothelial cells to retain the highly diffusible inflammatory chemokine for presentation to leukocytes.

In conclusion, these data show that, in addition to polarizing DC into mature cells that promote naïve T-cell differentiation into Th2 cells, histamine and PGE2 may act on immature DC to trigger local Th2 cell recruitment through a selective control of Th1/Th2-attracting chemokine production, thereby contributing to maintain a microenvironment favourable to persistent immunoglobulin E synthesis.

These results suggest that activation of members of the opioid and chemokine receptor families leads to downregulation of each other's leukocyte migratory activities.

Differential chemokine production by colonic epithelial cells is thought to contribute to the characteristic increased infiltration of selected population of leukocytes cells in inflammatory bowel disease.

We studied the clinical role of leukocyte infiltration and chemokine receptor expression in ovarian carcinoma effusions.

Adhesion molecules and chemokine signals function in concert to mediate this process and to organize leukocytes into distinct structures within the synovium.

Among the proinflammatory chemokine receptors, chemokine receptor (CCR)-1 has nonredundant roles for leukocyte adhesion to activated vascular endothelium and for transendothelial migration.

These results suggest that mammary epithelial cells are the source of chemokines in human milk and that the recruitment of leukocytes in human milk is likely to be chemokine-driven.

Chemokine [?] expression is markedly upregulated in healing myocardial infarcts and may play an important role in regulating leukocyte infiltration and activity and in modulating infarct angiogenesis as well as fibrous tissue deposition.

Chemokine induction mediates leukocyte recruitment in the myocardium.

Although CC chemokine receptors have been found predominantly on leukocytes, recent studies have suggested that vascular smooth muscle cells respond to CC chemokines.

The apparent recruitment of these leukocytes prompted us to search for chemokine expression by endometriosis cells.

Colorectal cancer (CRC) is characterized by a distinct metastatic pattern resembling chemokine-induced leukocyte trafficking.

CONCLUSIONS: These data demonstrate that chemokine expression is dysregulated in p-exposed endometria, consistent with the morphological appearance of the endometrium and the leukocyte subsets present.

The expression of chemokine receptors on blood and synovial fluid leukocytes was determined by 3-color flow cytometry analysis.

Leukocytes from chicken peripheral blood expressed chCXCR4 mRNA and responded to human SDF-1 in a calcium flux assay with an EC50 similar to that for chCXCR4-transfected CHO cells, suggesting that this response is mediated by native chCXCR4.

Altered leukocyte response to CXCL12 in patients with warts hypogammaglobulinemia, infections, myelokathexis (WHIM) syndrome.

We suggest that the **chemokine** binding to the ECs of lymphatics may be involved in the process of leukocyte entry into the afferent lymphatic vessels.

Since no significant correlation was found between csf leukocyte counts and **chemokine** concentrations or chemotactic activity mediated by csf, additional factors influence the extent of pleocytosis in vivo.

The **chemokine** signaling system, which coordinates the basal and emergency trafficking of leukocytes, presumably coevolved with the hematopoietic system.

Because chemokines facilitate the activation of leukocytes and their migration to the sites of inflammation, the modulation of **chemokine** production by the virus suggests a role for chemokines in immune evasion and/or immunopathogenesis of CMV retinitis.

Female gender attenuates cytokine and **chemokine** expression and leukocyte recruitment in experimental rodent abdominal aortic aneurysms.

Treatment with RWJ 67657 could lead to reduced leukocyte infiltration by the reduction of E-selectin expression and **chemokine** production.

The coordinated expression of cytokine and **chemokine** mRNA and protein was examined in various leukocyte populations and in inflammatory cells and fluid collected following the induction of tuberculous pleurisy in BCG-vaccinated guinea pigs.

Recently, T140 analogues have also been shown to inhibit CXCL12 [?]-induced migration of breast cancer cells, leukaemia T cells, pancreatic cancer cells, small cell lung cancer cells, chronic lymphocytic leukaemia B cells, pre-B acute lymphoblastic leukaemia cells and so on in vitro.

Andrograpanin, a compound isolated from anti-inflammatory traditional Chinese medicine Andrographis paniculata, enhances **chemokine** SDF-1alpha-induced leukocytes chemotaxis.

When examined using complementary DNA microarray, up-regulation of genes which are associated with DNA repair, detoxification, apoptosis, cell morphology, cell adhesion, and signal transduction was seen in CD4(+) T cells upon **SDF-1** exposure.

The redistribution of the **chemokine** receptors and adhesion molecules to opposite poles of the cell in response to a chemoattractant gradient may guide cell migration and cell-cell interactions during lymphoid cell trafficking in immune and inflammatory responses.

Conversely, neutropenia in children with myelokathexis is a result of leukocyte retention in the bone marrow because of the mutations of CXC **chemokine** receptor 4, which affect the capacity of cells to recirculate between blood and bone marrow.

Although understanding of the pathways leading to their development remains incomplete, data from in vitro studies suggest that neutrophils, monocytes, and their secreted products (eg, hydrogen peroxide, H2O2) influence the pathogenesis of pulmonary granulomatous disease through the regulation of local **chemokine** and cytokine production.

These observations represent the first detailed analysis of chemokine [?] production by lymphatic endothelial cells and may account, in part, for the mechanism of leucocyte recruitment into the lymphatics, and of lymphocyte recirculation within the lymphatic system.

Chronic ethanol inhibits CXC **chemokine** ligand 10 production in human A172 astroglia and astroglial-mediated leukocyte chemotaxis.

Many cell types including lymphocytes, macrophages, bronchial smooth muscle cells, endothelial cells and eosinophils, are able to produce this **chemokine**, predominantly after cytokine stimulation, however little is known about its expression in human skin in vivo.

Conclusions-PE with moderately severe pulmonary hypertension (PE2.0) resulted in selective RV dysfunction, which was associated with increased **chemokine** expression, and infiltration of both neutrophils and monocyte/macrophages, indicating that a robust immune response occurred with RV damage following experimental PE.

Conjugated linoleic acids have no effect on TNFalpha-induced adhesion molecule expression, U937 monocyte adhesion, and **chemokine** release in human aortic endothelial cells.

The **chemokine** stromal-derived factor-1alpha (SDF-1alpha) is an essential regulator of **hematopoiesis**, **lymphocyte** homing, pre-B-cell growth, and angiogenesis.

The extent of mucosal injury may reflect bacterial density, the variability of different strains of H. pylori to induce **chemokine** expression in **epithelial cells** and the **oxidative burst** in **neutrophils**.

We also found that in primary **lymphocytes**, **CXCL12 [?]** did not induce appreciable **phosphorylation** of any of the Jaks compared with cytokines for which these kinases are required.

A variety of control studies support the idea that infectious **EBV** is not required for induction of **chemokine** **gene expression**; however, the response is dependent on the interaction between the **glycoprotein** gp350 of the viral envelope and the **neutrophil** surface.

Expression of **chemokine** receptors on intrahepatic and peripheral **lymphocytes** in **chronic hepatitis** C infection: its relationship to liver inflammation.

Chemokines and **chemokine** receptors play important roles **in migration** and tissue localization of various **lymphocyte** subsets.

The expression of **chemokine** receptors on peripheral blood **lymphocytes** and thymocytes of **myasthenia gravis** (MG) patients was analyzed before and after therapy with special reference to the thymic histopathology.

Inhibition by PMA was reversed by co-treatment with GF109, implying that heterologous PKC activation is capable of desensitizing **chemokine** and **fMLP**-induced **monocyte** **chemotaxis**.

Characteristic for a **common cold** is the selective **neutrophil** recruitment and time-limited increase in mediator, cytokine, and **chemokine** concentrations that orchestrate **chemotaxis**, transmigration, and activation of inflammatory and immunocompetent cells.

Conclusion The results of this study suggest that **trophoblast** cells are able to recruit and successfully educate **monocytes** to produce and secrete a pro-inflammatory cytokine and **chemokine** profile supporting its growth and survival.

Thus, collagen XVIII may provide a link between **selectin**-mediated **cell adhesion** and **chemokine**-induced cellular activation and accelerate the progression of **leukocyte** infiltration in renal inflammation.

This work describes a previously uncharacterized mechanism for CC **chemokine** receptor down-modulation that is dependent upon **tyrosine** kinase activation and **serine** proteinase-mediated receptor degradation and may provide further insight into the mechanisms of **leukocyte** regulation during immunological and inflammatory responses.

How does the **lymphocyte** "choose" a direction for migration in the complex cytokine/**chemokine** environment of inflamed **dermis** when multiple chemokines secreted by multiple cell types are present?

Although often associated with **lymphocyte** recruitment, increased **chemokine [?]** expression is also associated with non-lymphocyte-mediated **CNS disease**.

These observations suggest a "push-pull" model, in which **lymphocyte** extravasation is driven by **lymphocyte** activation, expression of adhesion molecules, and increased **vascular permeability** and is coupled with **chemokine**-mediated trafficking to inflammatory sites in the CNS.

Renal **chemokine** expression in **immune complex disease** may thus be triggered as **lymphocytes** traffic through the kidney and encounter deposited immune complexes.

Conclusions: Our results confirm that SS is a Th2 disorder with a selective expression of CCR4, whereas inflammatory **erythroderma** shares an overexpression of both Th1- and Th2-related **chemokine** receptors, suggesting an activation of different pathways driving reactive **lymphocytes** to the skin.

In **alcoholic cirrhosis**, **chemokine** mRNA was detected in portal tract **endothelium**, **leukocytes**, and **fibroblasts**.

Lipocortin 1 and **chemokine** modulation of granulocyte and monocyte accumulation in experimental inflammation.

We investigated control of **chemokine** secretion from osteoblasts infected with either Mycobacterium tuberculosis, which normally elicits a granulomatous host response, or Staphylococcus aureus, which drives a host response dominated by neutrophil influx.

Biopsy neutrophilia, neutrophil **chemokine** and receptor gene expression in severe exacerbations of chronic obstructive pulmonary disease.

Distinct patterns of **chemokine** expression are associated with leukocyte recruitment in alcoholic hepatitis and alcoholic cirrhosis.

Chemokine expression is dysregulated in the endometrium of women using progestin-only contraceptives and correlates to elevated recruitment of distinct leukocyte populations.

We examined **chemokine** gene expression following the differentiation of a monocyte, macrophage cell lineage.

Chemokine profile of herniated intervertebral discs infiltrated with monocytes and macrophages.

5. These findings suggest that thalidomide and one of its derivatives impairs CD-like TNBS-induced colitis in the rat by down-regulating endothelial adhesion molecule and **chemokine** expression and, as a consequence, the interaction of these cells with circulating leukocytes.

Chemokine expression in human monocytes/macrophages exposed to Ti-alloy and PMMA particles in vitro was determined by RT-PCR, ELISA and monocyte migration.

Chemokine production has been associated with leukocyte infiltration into the joint during gouty arthritis, and monosodium urate (MSU) crystals, the causative agent of this arthropathy, have been shown to modulate their expression.

Suppressive activity of fexofenadine hydrochloride on thymus- and activation-regulated **chemokine** production from human peripheral blood leukocytes in response to antigenic stimulation in vitro.

The purpose of the present study was to assess a number of new chemokines for suppressive activity and to delve further into **SDF-1**-mediated chemotaxis of progenitor cells.

This strategy impacts key aspects of microbial pathogenicity as exemplified by increased bacterial invasion of epithelial cells and inhibition of **chemokine**-induced chemotaxis.

Our data provide a mechanism by which 2 **chemokine** gradients that are oriented in opposite directions could cooperate in efficiently driving out monocytes from blood vessels into tissue.

A blockade of complement activation renders the **chemokine** milieu unattractive to neutrophils and also modulates the alloimmune response toward Th2 cytokines, which may have an antiproliferative role in fibroproliferative disorders.

INTRODUCTION: Stromal cell-derived factor (SDF)-1 (CXC **chemokine** ligand-12) is a member of the CXC subfamily of chemokines, which, through its cognate receptor (CXC chemokine receptor [CXCR]4), plays an important role in chemotaxis of cancer cells and in tumour metastasis.

In addition, **SDF-1** is responsible for attracting mature lymphocytes to the bone marrow and can therefore contribute to host versus graft rejection in bone marrow transplantation.

In addition to lowering the plasma levels of Hcy, low-dose folic acid treatment exerts beneficial effects on patients with HHcy by inhibiting pro-inflammatory responses such as **chemokine** secretion from human monocytes.

The **chemokine** RANTES (Regulated upon Activation, Normal T Expressed and Secreted) attracts and activates primarily monocytes and may contribute to the pathogenesis of middle ear inflammation.

Chemokine RANTES is upregulated in monocytes from patients with hyperhomocysteinemia.

In mammary tissues **SDF-1** staining was primarily seen in **stromal cells** and weakly in mammary **epithelial cells**.

Cytokine/**chemokine** expression and surface expression of these novel cell surface receptors is dependent upon the **neutrophil** responding to local environmental factors to selectively up-regulate the expression of key cellular components via signalling pathways coupled to **transcriptional activation**.

RESULTS: **Tannic acid**, at nontoxic concentrations, specifically inhibited **CXCL12**-induced human **monocyte** migration (IC(50), 7.5 micro g/ml) but did not inhibit CCL2-, CCL3-, CCL5-, formylmethionylleucylphenylalanine (fMLP)-, or C5a-induced migration.

Differential effects of **9-cis retinoic acid** on expression of CC **chemokine** receptors in human **monocytes**.

The most potent compound discovered, namely (2S)-N-[3,5-bis(trifluoromethyl)benzyl]-2-cyclopropyl-4-[(1R,3'R)-3'-methyl-1'H-spiro[indene-1,4'-piperidin]-1'-yl]butanamide (29), showed very high binding affinity (**IC50** = 4 nM, human **monocyte**) and excellent selectivity toward other related **chemokine** receptors.

CONCLUSIONS: Overall, results from this study suggest that **homocysteine** alters the profile of cytokine/**chemokine** production by **endothelial cells** and **macrophages**.

Here, we report the ability of **homocysteine** to influence inflammatory cytokine/**chemokine** production by human **saphenous vein endothelial cells**, peripheral blood **monocytes** and **monocyte-derived macrophages**.

Our results show that **SDF-1** protein and mRNA are normally expressed by **endothelial cells**, **pericytes**, and either resident or explanted CD1a+ **dendritic cells**.

Increased levels of the **neutrophil** **chemokine** interleukin (IL)-8 in the lungs of severe trauma patients can predict subsequent development of **acute respiratory distress syndrome**.

Interleukin (IL)-8, the C-X-C **chemokine**, is a potent **neutrophil** chemoattractant that has been implicated in a number of inflammatory airway diseases such as **cystic fibrosis**.

Closed-eye tears were able to recruit **neutrophils**, with maximal recruitment after 8 hr of sleep, suggesting that **chemokine** IL-8 and the lipid chemoattractant **LTB4** were active.

Production of C-X-C dominant **chemokine** by **neutrophils** is consistent with the pathological characteristics of H. pylori-induced **gastritis**, where persistent **neutrophil** infiltration is present.

As prior work suggests that **thrombus** maturation involves early influx of **neutrophils** (PMN) and neovascularization, we hypothesized that administering the proinflammatory/proangiogenic **chemokine** interleukin (IL)-8 might accelerate **thrombus** resolution.

METHOD AND RESULTS: Primary human **monocytes** were treated with **LTB4** and the supernatant was analyzed for cytokine/**chemokine** production by an immuno-protein array.

Our findings suggest the activation of **neutrophils** and **monocytes** in the fetus during **preeclampsia** involving enhanced **chemokine** activation, possibly contributing to the fetal **morbidity** of this disorder.

Hypochlorite-modified LDL: chemotactic potential and **chemokine** induction in human **monocytes**.

There is considerable evidence from cellular studies showing that both **respiratory epithelium** and recruited **neutrophils** contribute to the **chemokine** response in paramyxovirus infection.

To understand how **neutrophils** are recruited to the lung in **pneumococcal pneumonia**, the ability of pneumococcal components to elicit the **chemokine** interleukin (IL)-8 from monolayers of cultured human type II cells was assessed.

Parallel induction of epithelial surface-associated **chemokine** and proteoglycan by **cellular hypoxia**: implications for **neutrophil** activation.

Antimacrophage **chemokine** treatment prevents **neutrophil** and macrophage influx in **hyperoxia**-exposed newborn rat lung.

These results indicate the ability of **erythromycin** to reduce CXC **chemokine** production and to enhance **neutrophil** degranulation in human blood.

Biologically active **neutrophil** **chemokine** pattern in **tonsillitis**.

In acute **sinusitis**, the synthesis of proinflammatory cytokines and of the **neutrophil** **chemokine** IL-8 and IL-3 appeared to be upregulated.

Our results suggest that early **B-cell** **lymphopoiesis** is important for **B-cell** recovery following rituximab, and that perturbation of **SDF-1** during **B-cell** recovery retards **neutrophil** egress from the **bone marrow**.

The ability of APC to decrease the release of the C-C **chemokine** MIP-1-alpha from the monocytic **cell line** THP-1 and from human **monocytes** may identify a novel immunomodulatory pathway by which APC exerts its anti-inflammatory action and may contribute to control the inflammatory response in **sepsis**.

Liver-infiltrating **lymphocytes** in end-stage **hepatitis C** virus: subsets, activation status, and **chemokine** receptor **phenotypes**.

CXCL12 was expressed in both **glioma** and **endothelial cells** as assessed by immunostaining of surgical brain sections.

MATERIALS AND METHODS: The expression of **chemokine** receptors in human brain microvascular **endothelial cells** (BMVEC) and **coronary artery** **endothelial cells** (CAEC) **in vitro** and cryostat sections of the heart tissue was determined by light and **confocal** **microscopy** and **flow cytometry** with monoclonal antibodies.

Damnacanthal, a specific Lck inhibitor, but not the Syk inhibitor **piceatannol**, inhibited **CXCL12**-induced **chemotaxis** of both **lymphocyte** subsets.

Expression of cytokine, **chemokine**, and adhesion molecules during **endothelial cell** activation induced by antibodies against **dengue** virus nonstructural protein 1.

We hypothesized that human (h)PrMCs and their mature counterparts might share overlapping patterns of **chemokine** and cytokine receptor utilization with **eosinophils**, **basophils**, and T helper type 2 (Th2) **lymphocytes** for their homing and allergy-associated **hyperplasia**.

The CC **chemokine** eotaxin has been characterized as an important mediator in **allergic reactions** because it selectively attracts **eosinophils**, Th2 **lymphocytes**, and **basophils**.

Lactobacilli and streptococci induce inflammatory **chemokine** production in human **macrophages** that stimulates **Th1 cell** **chemotaxis**.

APC0576, a novel inhibitor of NF-kappaB-dependent **gene activation**, prevents pro-inflammatory cytokine-induced **chemokine** production in human **endothelial cells**.

CXCL12 is expressed at the basolateral surface of CNS **endothelial cells** in normal **spinal cord** and at the onset of EAE.

Angiotensin II stimulates expression of the **chemokine** RANTES in rat glomerular **endothelial cells**. Role of the **angiotensin** type 2 receptor.

CONCLUSIONS: This study demonstrates that catecholamines differentially influence **chemokine** production and indicates that **DA** may have anti-inflammatory properties because it delays the expression of adhesion molecules and inhibits the production of chemokines in PTECs and **endothelial cells** under basal and inflammatory conditions.

Postcapillary **venule** **endothelial cells** in kidney express a multispecific **chemokine** receptor that is structurally and functionally identical to the erythroid isoform, which is the Duffy blood group antigen.

Regulation of **endothelial cell** branching **morphogenesis** by endogenous **chemokine** stromal-derived factor-1.

Prostate cancer cells were also observed migrating across **bone marrow** **endothelial cell** monolayers in response to **SDF-1**.

SDF-1 [?] also stimulated tube formation of human umbilical endothelial cells, and the response was LY294002-sensitive.

Characterization of a novel chemokine-containing storage granule in endothelial cells: evidence for preferential exocytosis mediated by protein kinase A and diacylglycerol.

The chemokine IL-8 is found on the luminal side of vascular endothelial cells, where it is postulated to be immobilized during inflammation.

SDF-1-alpha (50 ng/ml) increased the number of cultured endothelial cells from 33,653 +/- 1183 to 55,398 +/- 2741, which significantly reduced by adding the BK(Ca)-inhibitor iberiotoxin, or the endothelial nitric oxide synthase-blocker, L-NMMA (n = 24, p < 0.05).

CXC chemokine suppression of polymorphonuclear leukocytes apoptosis and preservation of function is oxidative stress independent.

We investigated the balance between Th1 and Th2 cells and between Th1- and Th2-associated chemokine receptor expression on peripheral lymphocytes in subjects including patients with coexisting type 1 diabetes and Graves' disease.

Anti-CD19 chimeric receptor-modified CIK cells became cytotoxic against B-ALL cells (mean lysis, 60%) and showed, after exposure to a CXCL12 gradient, high capacity to adhere and transmigrate through endothelial cells and to invade Matrigel.

Increased lysophosphatidylcholine and non-esterified fatty acid content in LDL induces chemokine release in endothelial cells. Relationship with electronegative LDL.

Here we used lymphatic endothelial cells (LEC) derived from experimentally induced murine lymphangiomas to investigate the pattern of chemokine [?] expression by these cells.

Respiratory syncytial virus (RSV) is an important cause of bronchiolitis in infants, is an important trigger of asthma exacerbation, and stimulates chemokine production by human respiratory epithelial cells in vitro.

We found that CXCL12 is expressed by bile duct epithelial cells in normal liver tissue.

Physiologically more important, L-selectin stimulation increased SDF-1-induced lymphocyte adhesion and transendothelial migration, which were inhibited by anti-leukocyte function-associated antigen 1 antibodies, tyrosine kinase inhibitors, and pertussis toxin.

Enhanced CXC chemokine responses of human colonic epithelial cells to locus of enterocyte effacement-negative shiga-toxigenic Escherichia coli.

In this study, it was investigated whether Physiomer, an isotonic sea water-derived solution commercialized for cleaning the nasal mucosa, impaired the chemokine IL-8 expression and secretion by human respiratory epithelial cells compared with that obtained with an isotonic 9% NaCl solution.

We investigated whether montelukast and zafirlukast could suppress chemokine-induced chemotaxis of monocytes and signaling.

The main cellular sources of chemokine mRNA were ductal epithelial cells and infiltrating mononuclear leukocytes.

Butyrate switches the pattern of chemokine secretion by intestinal epithelial cells through histone acetylation.

Human bronchial epithelial cells (HBECs), which can act as immune effector cells and express beta2-adrenoreceptors, were used to test the effects of different concentrations (0.1-100.0 nM) of salmeterol (Salm) on adhesion molecule expression and chemokine/cytokine release.

In the current study, we investigated the effect of IL-4 on chemokine production by human epithelial cells infected with Mycobacterium bovis bacillus calmette-guérin (BCG).

Quercetin blocks airway epithelial cell chemokine expression.

Epigallocatechin-3-gallate impairs chemokine production in human colon epithelial cell lines.

The aim of this study was to evaluate the direct immunoregulatory effect of **lidocaine** on pro-inflammatory cytokine and **chemokine** secretion from intestinal **epithelial cells**.

1 alpha,25-Dihydroxyvitamin D3 inhibits pro-inflammatory cytokine and **chemokine** expression in human corneal **epithelial cells** colonized with **Pseudomonas** aeruginosa.

Expression profile of chemokines and **chemokine** receptors in **epithelial cell** layers of **oral lichen planus**.

Distinct patterns of expression of each **chemokine [?]** were noted on **Kupffer cells**, sinusoidal **endothelial cells**, **hepatocytes**, **lymphocytes**, and **bile ducts**.

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